b.—PATHOLOGY OF THE NERVOUS SYSTEM AND MIND, AND PATHOLOGICAL ANATOMY.

SPINAL DISEASE FROM RAILROAD TRANSIT.—It seems to be highly probable that continuous railroad traveling has an injurious effect on the nervous system, especially among those railroad employes who, in the course of their duties, are obliged to be constantly on their feet while the cars are in motion. Dr. W. J. Scott, of Cleveland, Ohio, contributes to the *Med. & Surg. Reporter*, Oct. 2, 1875, an account of several cases in his own observation of what he calls "railroad locomotor ataxia." He says these are only a portion of the cases he has observed; others could be added. They are undoubtedly cases of spinal disease, though as described some of them may have been merely congestion or incipient myelitis rather than true locomotor ataxia—co-ordination, however, was impaired in all.

The explanation given by Dr. Scott appears rational, and is the same in the main as has been proposed by other authors. The constant tax on the spinal centres to maintain equilibrium in the constantly and extremely rapidly varying motions of the car or engine, may readily be supposed to produce conditions which might end in actual disease. Further observations in regard to this question are desirable.

ACUTE SPINAL PARALYSIS.—Dr. Leyden, of Strasburg, read before the German alienists at Heppenheim, in May last, a paper on this disease, an abstract of which as given in the *Allg. Zeitschr. f. Psychiatrie*, is as follows:

The name acute spinal paralysis, introduced by Duchenne, indicates, strictly taken, only an acute paralysis, in regard to which one is justified in thinking that it depends on a spinal trouble without being able to say the special form of disease. Just as the designation progressive spinal paralysis had to be given up for the chronic form as the pathological anatomy became better known, so presumably will it be for the acute spinal paralysis. According to Duchenne the disease in question is distinguished by the fact that it is an acute or subacute spinal paralysis, which is preferably or exclusively limited to the motor apparatus, readily advancing to muscular atrophy, is often curable, and commonly leaves muscular atrophies behind it. All other symptoms are inconstant. The difference between acute spinal paralysis and acute softening, is only one of degree. This last, a severe disorder, which sometimes comes on without any appreciable prodromata, depends on a disintegration of the cord which seldom permits any notable improvement; in acute spinal paralysis, on the other hand, the progress may be retrograde and allow a more or less perfect cure. If it is progressive it is not the intensity of the paralysis to the implication of the sphincters, etc.

which causes death, but its propagation into the medulla oblongata. We can recognize two kinds of acute spinal paralysis, differing in their course: one a multiple form (limited to one or several small patches of the cord) which corresponds to the acute spinal paralysis of adults; and a progressive form, the type of which is the acute ascending paralysis of Laudry, but which can also be descending, and which is fatal through implication and paralysis of the medulla oblongata.

Up to the present time we have very few anatomical data as to this disease; the autopsies commonly afford nothing. In the published observations of French writers, in two cases of acute ascending paralysis there was found an acute atrophy of the ganglion cells, without any other inflammatory condition in the cord. An albuminous substance filled the ganglion cells and surrounded the vessels. A special atrophy is not indicated here, and Leyden is of the opinion that a progressive degeneration of the ganglion cells does not at least lie at the base of the primary process.

Leyden himself has twice found a limited local spinal lesion, analogous to that in infantile paralysis in adults. He believes that at least a part of the cases of the progressive form depend on a lesion, which originates in the medulla; and the symptoms of œsophageal paralysis observed in the beginning by most authors favor this view. The process is either an ascending one which secondarily attacks the medulla, or it originates there. In several cases it seemed to be connected with a disease in the vicinity of the central canal, a *sclerose peri-ependymaire hallopeaus*. The central canal itself seemed gaping, or in its neighborhood are fissures produced by a degeneration of its surrounding substance, which was in part sclerosed, in part softened. (Drawings of these appearances were exhibited.)

The lately observed case of the author was as follows: A woman, forty years old, powerfully built, and always before healthy. All at once she felt a jerk and a prickling in the right side; the sensibility was undisturbed, the motility seemed diminished in all four extremities; the following day œsophageal paralysis made its appearance, and in this condition she was brought into the clinic, where for several days she had to be fed with the stomach tube. Besides the paralysis of the œsophagus, hoarseness gradually made its appearance, ending in complete aphonia, while the tongue still remained easily movable. The face was slightly drawn to the right, the right pupil dilated. Death occurred from paralysis of the respiration; the patient was strongly cyanosed. The symptoms did not correspond perfectly with those of the typical form of acute ascending paralysis, still we can include the case under that head, since it began and continued acute, and ended in asphyxia.

The post-mortem revealed in the freshly prepared medulla no abnormality; in the cervical cord was found a fissure which corresponded in situation to a dilated central canal. On the other hand, in the hardened preparation there was found a limited lesion on the right side of the medulla, which gave ample reason for the symptoms observed during life. The lesion did not include in its extent the nucleus of the hypoglossus, but did involve those of the glosso-pharyngeus, the accessorius and the vagus, and partly also that of the facial nerve. The diseased patch of the medulla was found

richly infiltrated with cells of almost an epithelial character, similar in form to granular cells but without any fatty degeneration; these cells are produced from the elements of the neuroglia. The patch was compact, showed no disintegration, and to microscopic examination showed only a commencing softening on the margin. That this patch is connected with the sclerosis of the central canal, the author holds as very probable, but not absolutely certain.

The second case was a woman about sixty years old, who died of dropsy and tuberculosis, and in whom paralytic symptoms appeared without their taking on any very characteristic course. The spinal cord showed pericentral sclerosis with cystic softening, most pronounced in the cervical region; the surrounding substance of the posterior and lateral columns showed a parenchymatous myelitic disease without softening, which extended upward and might easily have given rise to the symptoms of ascending paralysis; this myelitis was undoubtedly secondary to the pericentral sclerosis.

Hereditary Ataxia.—Friedreich, in a paper read at the meeting of German Alienists, at Heppenheim, in May last, (reported in the *Allg. Zeitschr. f. Psychiatrie*,) called attention to the fact that he had already, thirteen years previously, published (*Virchow's Archiv*, XXVI. and XXVII.,) a series of cases of spinal disorder, which, under the form of ataxia, were characterized by the following peculiarities: All cases depend on heredity; this affection occurs usually in young females; the outbreak of the disorder is always at the commencement of the sexual development of puberty; the course of the disease is slow with intervals of years; exacerbations and remissions occur; the onset is spontaneous, and in the later stages there always exist disorders of articulation, which are to be considered as ataxic; when the disease is at its height only disturbances of co-ordination, never paralysis, are observed; the disease ends with the appearance of ataxic disorder of articulation, and disorders of the cutaneous and muscular sensibility are never observed.

This form of disease, of which the author has observed six cases, he thinks should be distinguished from tabes dorsalis. He had had these cases under observation in the University hospital for the greater part of a year; three had died in the meantime from ilio-typhus. The autopsy showed in all three degenerative atrophy of the posterior spinal columns, and in all the lesion ceased at the base of the fourth ventricle, so that both the clinical symptoms and the anatomical results of the section are alike clear and precise.

This disorder seems to Friedreich to be somewhat rare, since he finds no mention of it in the literature, and for a long time he failed to observe any cases himself. Only in the last year he has had the good fortune, however, to discover a new family in which this disease is hereditary in three sisters, beginning in all in their thirteenth year and taking the same course as in those previously described. In one of the three, however, commencement of the disease was not as gradual as in the others, though its progress was precisely similar; the disease, as well as could be remembered, extended itself at once to both upper and lower extremities. The first sister died at home

and an autopsy was not allowed; the second is now in the care of her friends, and the third, temporarily in the clinical hospital at Heidelberg, allowed him permission to present her in person at the meeting.

The girl, now seventeen years old, has been affected for four years. Standing and walking are still possible without assistance. The speech is implicated in the ataxia. Irritations were promptly localized, and the patient was able to pick up an object from the table. The ataxic symptoms were not more pronounced with the eyes closed or when walking backwards. Trophic disorders were at no time present, and the cutaneous sensibility and the muscular senses were perfectly retained.

In view of these cases, in which no disorders of sensibility were at any time present, Dr. Friedreich cannot agree with the opinion which makes the ataxia a result of disordered sensibility, but agrees with Todd and Gull that the ataxia is perfectly independent of sensibility, and that it is purely the consequence of a disturbance in the co-ordinating centre, or those parts of the cord which mediate farther on in co-ordination (routes of the posterior columns). The co-ordination centre is doubtless to be located in the cerebellum; among other facts, those co-ordinatory disorders which result from tumors in this organ support this view. Thus we have two forms of ataxia to distinguish: 1, a cerebral or rather a cerebellar form, and, 2 a spinal form. Those cases described by the author belong to the spinal ataxias.

Cerebral Hæmorrhages in Infants.—J. Parrot, *Archives de Tocologie*, 1875 (abstracted by L. E. Dupuy, in *Rev. des Sciences Medicales*).

Cerebral hæmorrhages in the newly-born are distinguished by special characters altogether dissimilar from those observed in other cases: this study of M. Parrot's forms an entirely new chapter in the still very incomplete description it gives of the affections of the cerebro-spinal apparatus in young infants.

The author has personally collected the accounts of thirty-four cases of cerebral hæmorrhages. He notes that the clot has a marked preference for the lower parts; that it is most frequently bilateral, and that in cases where it exists on one side only, the right is more commonly affected than the left. If we add that the clots are generally situated on the surface of the convolutions, the sub-arachnoideal region, or the sub-ependyma of the lateral ventricles, and that they occur without any previous symptom permitting their diagnosis during life, we can readily see how this form of hæmorrhage differs in all points from the same affection observed later in life.

M. Parrot has sought to discover what might be the primary causes of cerebral hæmorrhage in the newly-born. His observations have demonstrated to him that it is produced in the course of other diseases, which are thus enumerated in the order of their observed frequency as causes of this accident: Arthrepsy, 24 times; Œdema neonatorum, 8 times; General Congestion of the viscera, 1 case; Hypertrophy of the thymus, 1 case.

Therefore in the great majority of cases the hæmorrhage is produced during the evolution of arthrepsy and is caused by the disorders of nutrition which constitute that affection.

One of the first effects of arthrepsy is to cause an alteration of the blood, which finally stagnates or even coagulates in the veins; hence results a venous congestion of most of the viscera and especially of the periphery of the encephalon.

On the other hand the cephalo-rachidian liquid is considerably diminished by these same nutritive disorders, and ceases to protect the vessels of the brain, which are found pressed against the hard surfaces of the cranial bones. These facts sufficiently explain the origin of the cerebral hæmorrhages in young infants.

The constant appearance of the clots in the lower and posterior portions of the brain is due to the dorsal decubitus which is the habitual position of infants in the hospitals. As regards the considerable predominance of peripheral hæmorrhages over those in the nerve substance, it is necessary to attribute it to the fact that the central vessels suffer less than those of the periphery from the absence of the cephalo-rachidian fluid.

EXOPHTHALMIC GOITRE AND VITILIGO.—N. Raynaud, *Archives Generales de Medicine*, June, 1875 (abstracted in *Rev. des Sci. Med.*).

Trousseau, without attaching to it any special importance, has mentioned in one of his observations, the existence of a vitiligo in a patient suffering from exophthalmic goitre. (*Clin. Med.*, t. II.) The author reports three observations in which this singular coincidence has been remarked, and adds another from M. Delasiauve. (*Gaz. des Hopitaux*, 1874, p. 1157.) The patches of vitiligo have no special seat, they may exist on all parts of the body and are either discrete or even confluent. As to the pathogeny of this coincidence it is thus far unexplained.

SATURNINE HEMIANÆSTHESIA.—A. de Conos, *Thesis de Paris*, 1875, (abstracted in *Rev. des Sci. Med.*), offers the following conclusions in regard to the symptom of hemianæsthesia in cases of lead poisoning:

1. Hemianæsthesia may occur, under certain rare circumstances, as a symptom of saturnine intoxication.

2. It approaches hysterical hemianæsthesia and that of cerebral origin in its general characters, but differs in some special features. Thus it always appears after a series of prodromata; its *debut* is slow and progressive; its progress gradual; and we do not observe before its establishment any of the precursory phenomena of hæmorrhage or cerebral softening. Finally we observe sensible differences in the manner in which insensibility to electric action is distributed in the patient.

3. Saturnine hemianæsthesia has probably its point of departure in an alteration of the encephalon, but the lesion which produces it is unknown.

4. Its prognosis is favorable.

THE IDIOPATHIC THROMBOSIS OF THE CEREBRAL ARTERIES AS A CAUSE OF HEMIPLEGIA IN TUBERCULAR BASILAR MENINGITIS.—Ludolph Schuh, *Verhandl. der Wurzb. phys. med. Gesell.*, VIII, 179 (abstracted in *Rev. des Sci. Med.*)

In tubercular basilar meningitis the troubles of motility may have three separate causes: 1st, tubercles of the brain; 2d, white softening; 3d, thromboses of the cerebral vessels.

The first two of the complications of tubercular basilar meningitis are rather common, and the author cites cases in which we can very easily fix the relation between the motor disorders and the cerebral affection.

The third is much less known; the author cites three cases, two from the Thesis of Ferber (Leipzig, 1861,) and one from Jenner (*Schmidt's Jahrb.* 109, p. 100).

The conclusions he draws from these facts are as follows:

1. The idiopathic thrombosis of the brain presents a prodromic period like that in other cerebral affections.

2. The morbid symptoms become more and more pronounced as the thrombosis becomes more decided.

3. The attack itself resembles an attack of apoplexy, but later we see an augmentation or rather the persistence of the paralysis, while apoplexy is followed by the diminution or the cure of the paralytic symptoms.

4. We find very often a red softening in the side of the brain opposite the paralysis, ordinarily hemiplegic. This red softening is nevertheless not constant.

5. In the majority of cases the patient preserves his consciousness during the attack.

6. The prognosis is very unfavorable; we find in the literature only one case of cure.

As to the mode of production of the thrombosis, the author admits that the tubercle, first situated on the vascular walls, tends more and more to penetrate into the circulatory torrent. The heart, always weakened in the subjects of tubercular meningitis, can only afford a feeble impulsion against this invasion of the vessels and the deposit of the tuberculous masses.

PATHOLOGY OF THE PNEUMOGASTRIC.—Dr. S. O. Habershon, *Guy's Hospital Rep.*, XX., 1875, 127 (abstracted in *Rev. des Sci. Med.*), after some practical conclusions on the anatomy of the vagus, narrates several observations, from which he deduces these three general propositions:

1. A morbid alteration located at the cerebral origin of the vagus may produce symptoms of irritation in all parts supplied by this nerve.

2. An irritation of a certain number of peripheral branches may cause disturbance in every part supplied by the nerve and also in the nervous centre itself.

3. Alternate irritations may appear sometimes on one branch, sometimes on another.

The fourth case of Dr. Habershon, detailed in this paper, is called up in support of the third of the above propositions. In this account we see a young man, twenty-four years of age, presenting alternately asthma, cardiac palpitations, cephalalgia, and gastric disorders.

Rheumatic Alterations of Muscular Nerves.—Richter, *Deutsches Archiv f. Klin. Medicine*, XV., 368 (abstracted in *Revue des Sci. Med.*). "In muscular rheumatism the affection does not consist primarily in a hyperæmic or inflammatory process, but in a neurosis of the muscular nerves which manifests itself by pain, cramp, and paralysis. The morbid state of the muscular nerves has for its result, partly hyperæmia from vaso-motor paralysis, partly inflammation of the muscular tissue. This hyperæmic and inflammatory process exercises a destructive action on the nervous apparatus, already diseased, of the muscle, and may produce atrophy. The excitation of the sympathetic may also, by itself, cause trophic disorders."

Skin Disorders in Progressive Muscular Atrophy. — H. Balmer, *Archiv der Heilkunde*, XVI., 4, 1875, after reviewing the theories as to the pathology of progressive muscular atrophy, publishes the accounts of three cases, two of them taken from the monograph of Friedreich and one of his own observation, in which the disorder was complicated with lesions of the skin, vesiculæ, excoriations, alterations of nails, etc., and also congestions of the skin, profuse local perspirations and other disturbances. These phenomena are attributed by the writer to the same cause as zoster, pemphigus, etc.: an alteration in the vaso-motor nerves and their centres. This same disorder of the sympathetic nerves and centres acts also on the muscles, as well as on the skin, causing atrophy of the one and eruptions of various kinds on the other.

Without holding that all cases of progressive muscular atrophy are dependent on disordered function of the sympathetic, Dr. Balmer is inclined to believe that those in which there co-exist pronounced lesions of the skin are connected with disorders of the main trunks or ganglia of the sympathetic, or disturbance involving the vaso-motor and trophic centres.

Hydrophobia.—At the last session of the medical section of the French Association, at Nantes, in August last (reported in *Revue Scientifique*), M. Masfranc maintained that dissatisfied sexual desires were the cause of hydrophobia in dogs, and as a corollary to this theory, so often advanced and disputed, he advised the preservation of a larger number of bitches as a prophylactic measure.

BULBAR PARALYSIS.—Dr. Hallopeau, *Thesis d'Agregation*, Paris, 1873 (abstracted in *Rev. des Sci. Med.*).

After a short exposition of the anatomy and the normal physiology of the medulla, the author, in the chapter entitled pathological physiology, begins the study and the physiological interpretation of the principal symptoms met with, or that may be met with, in the affections of the medulla (direct and crossed or alternate paralyses, amyotrophies, contractures, disorders of the circulation, the respiration, the salivary secretion and the urine); this chapter, in a word, is an attempt at localization of the bulbar paralyses.

Commencing next on the chemical side, properly speaking, of the question, M. Hallopeau starts with a description of the best known bulbar paralysis, that which has been described in so masterly a manner by M. Duchenne, under the name of glosso-labio-laryngeal paralysis. Thanks to the anatomico-pathological researches of M. Charcot and his students, we are able to-day to specify the lesion which produces this curious syndrome, and which consists in a chronic primary atrophy of the motor nuclei in the medulla. In Duchenne's disease the medulla, and it alone, becomes the locality of a lesion identical with that which occurs in the anterior horns of the cord in progressive muscular atrophy.

There exist, however, forms very different from that described so clearly by Duchenne. To the paralysis of the muscles innervated from the nuclei of the medulla may be added peripheral paralyses; the disease may commence with the medullary symptoms, or it may begin at the extremities under the form of progressive muscular atrophy, which is finally complicated with glosso-labio-laryngeal paralysis; it is this which M. Hallopeau calls the mixed form, or the spino-bulbar type.

We are aware that M. Duchenne, resting himself on the fact that he had never seen the atrophy of the muscles affected in glosso-labio-laryngeal paralysis, refused to consider this disease, with M. Charcot, as a simple bulbar localization of a lesion identical in its nature with that of progressive muscular atrophy. After a thorough discussion M. Hallopeau refutes this distinction.

In the preceding diseases the motor bulbar nuclei are seized protopathically; they can, however, be affected secondarily to inflammatory or sclerotic lesions of the other parts of the medulla. This is what we observe in lateral amyotrophic sclerosis (Charcot), when the bulbar symptoms are constant; the manner of their appearance is variable—most frequently they appear at the termination, but sometimes at the beginning of the disease.

In general spinal paralysis bulbar symptoms may present themselves. (Cases of Duchenne, Cornil and Lepine.)

The troubles of speech, the tremors of the lips and tongue, the hindrance of deglutition, the cardio-pulmonary accidents of multiple sclerosis, are all symptoms of the localization of the disease in the medulla.

Lesions *en foyer* (hæmorrhage, softening), exclusively limited to the medulla are rare, especially in cases which are subjected to post mortem examinations; the clinical tableau is that of Duchenne's disease, but with an abrupt commencement, habitually without loss of consciousness; with or without

paralysis of the members; the amelioration, when it occurs, is rapid and may be definitive; frequently the patient has a relapse; it may be rapidly fatal. The author insists upon these reservations in the diagnosis.

Only seven cases of tumor limited to the medulla have been reported; according to the analysis of the various symptoms observed, the author concludes that it is impossible to write a correct general history of the symptomatic paralyses from tumors of the medulla.

Finally, under the head of bulbar paralyses without determinate lesions, M. Hallopeau enumerates various forms of paralyses (diphtheritic, saturnine, hysterical) which may be localized in the medulla, but of which it has thus far been impossible to specify the lesion.

A final and interesting chapter is given to the symptomatology of bulbar paralyses, but these generalities do not admit of any analysis.

THE RELATIONS BETWEEN EPILEPTIC SYMPTOMS AND AURAL DISORDERS.—Dr. Moos, of Heidelberg, presented a paper to the meeting of the Southwestern German Alienists, at Heppenheim, May 1 and 2, 1875, on the connection between epileptiform symptoms and diseases of the ear, of which we take the following abstract from the *Allg. Zeitschrift f. Psychiatrie*, XXXII., 5.

The speaker gave first a historical sketch of the literature of the more simple and the more complicated neuroses, which are sometimes connected with disorders of the outer and sometimes with those of the inner ear; described the convulsive attacks of sneezing, coughing and vomiting in such cases, and expressed the opinion that, with the aid of this material, the more complicated reflex disturbances might be classified under three heads: reflex epilepsies, reflex hemiplegias, and reflex psychoses; the two first of these being sometimes observed to co-exist.

1. Reflex epilepsy. To this head belong the cases reported by Fabricius, Heldanus and Maclayan—foreign bodies in the outer ear. Those of Köppe and Schwarze, as well as one of the speaker's own cases—purulent inflammation in the middle ear, with partly epileptic and partly tetanic accidents.

2. Reflex hemiplegia. In this category come the cases of Hillairet and Handfield Jones. Reflex epilepsy co-existed. In these cases insects found their way into the external auditory passages, producing the irritative phenomena.

3. Reflex psychoses. Two cases of Köppe.

Dr. Moos gave a detailed account of the case of reflex epilepsy observed by him. The patient was a boy eight years old; the attacks occurred every four to eight weeks, and lasted two or three hours; were attended with loss of consciousness, and fixed staring countenance, but without convulsions; they ended with an attack of vomiting. The Eustachian tube of the right ear being found impervious the tympanum was twice incised before the ear trouble and the accompanying neurosis could be cured.

Dr. Moos explains the epileptic symptoms as follows:

The catarrh of the middle ear gradually progressed until it came to the point of purulent inflammation. This produced an irritative condition in

the tympanic plexus, which centripetally propagated itself to the brain, and with the assistance of a hereditary predisposition—(a 14-year old brother of the patient is weakminded, and a maternal uncle "suffered from the falling sickness, and died in his twenty-eighth year of tuberculosis")—produced alterations in the brain which manifested themselves peripherally in these epileptic phenomena.

The case is suggestive and important, not only to the neuropathologist but also the general physician, since ear troubles and convulsions are frequent in children, and the starting-point of many cases of convulsions is not yet known.

Syphilitic Lesions of the Cerebral Arteries.—The following are the conclusions of a recent *brochure* by M. Rabot, as reported in the *Gaz. des Hopitaux*, No. 115, 1875:

Tertiary syphilis may act on the arteries as on other organs. It gives rise to the formation of gummata. Thus far we need not admit the presence of endarteritis, but we would not be as positive in regard to periarteritis. The results of tertiary syphilis on the cerebral arteries is softening, of which the symptoms vary very little from those of softening, which has no connection with this disease.

Influence of Section of the Trigeminus of the Cornea.—The following are the conclusions of an article by Dr. Senftleben, in *Virchow's Archiv*, LXV., 69, on this subject:

1. The affections of the cornea resulting from section of the trigeminus are independent of any influence of trophic nerve fibres, since, probably, none of these exist in that nerve.

2. The primary corneal affection from section of the trigeminus is a necrosis, caused by repeated injuries which are the result of the consequent anæsthesia of the eye.

3. The circumscribed necrosis of the cornea acts as an inflammatory irritation, and produces a secondary inflammation of the cornea advancing from the periphery.

4. The simultaneous extirpation of the superior cervical ganglion of the sympathetic has no influence on the production and the subsequent course of the affection of the cornea following section of the fifth nerve.

Vertigo and Sea-Sickness.—At the meeting of the Paris Academie de Medicine, reported in *Bull. Gen. de Therapeutique*, Dr. Piorry read a long paper on vertigo, which he ranked among the neuropathies; he specially mentioned sea-sickness, which he considered an irisalgia; those persons, therefore, who are subject to it should avoid fixing their attention for any long time consecutively on objects that actively stimulate the eyes. This

precept is especially applicable to patients who present any degree of strabismus. They should stay extended in the horizontal position and in the darkness also, to overcome the optical sensation they feel, and they should call in all the power of the will to allay the inquietude they may feel. As to the treatment of vertigo and of migraine, M. Piorry advises the following:

Pressures, frictions of the affected eye, the application of warm bodies (and perhaps also, cold,) slight douches, or rather the projection of water on the lids, may be useful. We may even have recourse to the extract of belladonna, which, spread upon a substance which we apply to the diseased eye, promptly dilates the pupil; it is also well to use frictions on the lids, with some liniment containing morphine, etc.

A very simple and common hygienic treatment, which prevents sometimes the attacks of migraine, consists in provoking, at the beginning, the physiological action of the stomach by taking a few spoonfuls of good wine and biscuit; this is very useful in irisalgia, at the first appearance of the irisalgic circle.

It is likewise important to combat the complications or the circumstances which predispose to vertigo, the dyspepsias, for example (*vertigo a stomaco*). We give in these cases bicarbonate of soda, in large doses, and bismuth. When there is tumefaction of the stomach we give extract of berberis or alcohol of quinine. In plethoric individuals we practice the withdrawal of blood; for anæmic persons we prescribe reparative aliments, exercise, iron, etc.

M. Le Roy de Mericourt called M. Piorry's attention to the fact that sea-sickness may occur independently of any visual disorder. It is seen, often in its highest degree, in the most profound darkness, during sleep, and even in persons that are totally blind. Moreover, certain observations show that the intensity of sea-sickness is not in proportion to the amount of visual disturbance caused by the greater or lesser rapidity of the movements of the ship or the vessel on which the observer is placed.

Pathogeny of Neuralgias.—At one of the meetings of the medical section of the French Association for the Advancement of Science, in August last, M. Tripier made a communication on the pathogenesis of neuralgias, which is thus reported in the *Progres Medical:*

M. Tripier commenced by establishing the existence of the painful valleix points; then he demanded how the appearance of these points is to be explained.

He believed it could be done by admitting the recurrent fibres which terminated in the parts where this pain was felt. In fact, this hypothesis accorded with the law of Muller, who held that all sensations were perceived in the region of distribution of the nerves, at the termination of their filaments. M. Tripier cited cases where the pain felt in a nerve was calmed by pressure on an adjoining nerve trunk, the one which, according to this hypothesis, furnished the diseased recurrent fibres. M. Tripier likewise believed that he could explain by this theory of recurrent fibres, the

facts of the extension of neuralgias from one nerve to another one adjacent. He much preferred this assumption to that which made the lesion pass by way of the centres to the nerve thus secondarily affected. Finally M. Tripier said that the operation of section should be reserved for very obstinate neuralgias. He mentioned that Graefe had succeeded empirically in curing frontal neuralgia by dividing a large number of nervous fibres in the neighborhood of the painful point.

If we increase the pain at a neuralgic point by pressure over the nerve where it leaves a bony canal or a foramen, we may consider the diseased fibres as recurrent ones. If we wish to precisely locate the nerve which furnishes the diseased recurrent fibres, it is necessary to compress the neighboring nerves; if by pressure of one of these the pain is arrested, we may be sure that it is the one which supplies them.

The following are some additional articles, recently published, on the pathology of the nervous system and mind :

BEARDSLEY, Phimosal Paraplegia, *Med. and Surg. Reporter*, Aug. 21 ; WOOD, Diagnosis of the Lesion in Paraplegia, *Phil. Med. Times*, Oct. 2 ; BRUNET, Contagion of Insanity, *Ann. Med. Psychologiques*, Nov. 1875 ; SEMAL, The Alterations of General Sensibility in Melancholic Affections, *Ibid ;* GALLOPAIN, Interesting Observations of Softening of the Corpora Striata, *Ibid ;* OSGOOD, Angina Pectoris, *Am. Jour. of Med. Science*, October, 1875 ; LORING, Remarks on the Ætiology of Choked Disk in Brain Disease, *Ibid ;* FOURNIER, Tertiary Syphilitic Epilepsy, *L'Union Medicale* (cont. art.); LEGRAND DU SAULLE, The Insanity of Doubt, with Delusions of Touch, *Gaz. des Hopitaux* (cont. art.); BOUCHUT, The Differential Ophthalmoscopic Symptoms of Commotion and Compression of the Brain, *Ibid*, Oct. 19 ; LANGE, Remarks on Neuralgia and its Treatment, *Hospitale Tidende* (cont. art.); LEVINSTEIN, The Morphia Habit, *Berliner Klin. Wochenschrift*, Nov. 29 ; WILLIGK, Anatomical Findings in Cases of Cerebral Concussion, *Prager Vierteljahrschr.* XXXII., 1875, 4; KOLACZEK, On the Pseudo-Hypertrophy of Muscles, *Deutsche Klin. Wochenschr.*, No. 5, Oct. 23; PAULI, On a Psychic Symptom of Disorders of the Urogenital System, *Ibid*, No. 6, Oct. 30.